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A PATHOLOGICAL CONDITION OF THE LUNGS, HITHERTO UNDESCRIPTED IN THIS COUNTRY, BUT WHICH IS NOT INFREQUENT.

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DURING the course of a very prolonged service in hospitals I have repeatedly observed a condition of the lungs which is markedly distinct and characteristic, which I have not seen described. I speak of it at present only as a pathological condition, leaving it to others to decide whether it should take rank as a distinct disease.

Certain patients presented the following symptoms: Dulness, or sub dulness, generally at the middle, lateral, or posterior portions of the chest; there was always imperfect respiration; scarcely any râles were present, or if so, they were sparsely disseminated, and generally the subcrepitant; or, perhaps, in lieu of râles there was only rough breathing. The condition was consequent on antecedent morbid states, and there was not necessarily elevation of temperature; there did not exist evidence of any acute inflammation, or any of the well-known classic diseases of the chest—no pleurisy, pneumonia, bronchitis, pleurisy, emphysema, hydrothorax, etc. The positive physical signs of these several diseases were all absent—there was no crepitant, or sibilant, or crackling râles; neither was there pain nor rubbing sounds. So all the diseases which these signs indicated had to be excluded, and a name was wanted for the new condition which had been isolated.

But to continue the citation of positive and negative symptoms: The respiratory murmur, though not normal, was not completely absent, for the lung was still pervious to air; the vocal resonance—or what I prefer to term the reverberation of the voice—was slightly affected; some complementary respiration might be present, but this was not very decided, because there was no absolute consolidation; scarcely any dyspnoea may exist, and the cough may be moderate or absent.

Constantly, also, when an autopsy was afforded in such cases, the physical evidences of the diseases above cited were absent, and there was invariably present a large amount of bloody serum exuding from the cut surfaces, and it would flow most freely when the lung was squeezed. Here was plainly a gross morbid fact, which had to be noted and accounted for.

No disease of the heart coexisted, to the obstruction produced by which might be ascribed the collection of bloody serum; and though I have not yet been able to associate the two, I would not be surprised if cases were found to be dependent upon diseases of the heart.

In a hepatized or in a solidified lung the dulness is complete, and there is bronchial, and often puerile, respiration in the healthy areas, so that hepatization and solidification were also excluded. It was not oedema, for this is general over both lungs, and Lænnec had taught us that the crepitant râle is as characteristic of oedema as it is of pneumonia. I have heard, as it were, a shower of fine crepitation over the entire lungs in a case of oedema consequent upon measles; the oedema was detected by means of the râles. The crepitant râle was not pres-

ent, nor the fever, etc., essential to pneumonia; neither was there any rusty-colored sputa.

The conditions with which our cases would be most likely to be confounded would be the hypostatic congestion, or the hypostatic pneumonia of recent authors—if such a disease exists. But these will be referred to again.

I long since began to designate the condition, or disease, referred to as "Engorgement of the Lungs"—serum being always mixed with blood. I was compelled to use these terms, because they were true and applicable, and were essential in describing and interpreting the condition. I felt satisfied that others must frequently encounter a like pathological state, especially in hospitals; but I could get no response or support from authorities in this country. I wrote to Professor Austin Flint upon the subject years ago. The loss of his letter is regretted, together with the notes of cases with autopsies, though I possess the record of one observed in 1879, and had been observing and demonstrating the condition long anterior to that period. Some illustrative cases will be inserted at the conclusion of this paper.

My cases of engorgement of the lungs exist for days and weeks, and do not depend wholly upon the accidents of position, stasis of the blood, age of patient, or want of vitality. The powers of life are not specially enfeebled. They do not seem to depend on Bright's disease, dropsy after scarlet fever, measles, infectious diseases, etc.—as is the case with congestion of the lungs and oedema. I did not for a moment dream of calling them hypostatic congestion—certainly not in the old sense of the use of the term, for the condition is not necessarily hypostatic in cause.

The term infiltration is not very inapplicable, and the diagnosis may be attended with difficulty; but I have seen lungs infiltrated after severe pneumonias, and tubercular infiltrations exist; but in neither case is there often a large flow of serous fluid when the lung is cut, as is constant in engorgement of the lungs. This I have repeatedly observed in autopsies—some of them quite recent, and examined with a view to determining this question. Yet I have also recently seen serum with bloody and purulent matter exuding from the lungs in a well-marked case of general tuberculosis. The presence of fever, purulent expectoration, rise of evening temperature, and other signs of tuberculosis must be used to aid in their distinction from engorgement of the lungs.

I consulted, almost accidentally (1888), Ziemssen's "Cyclopædia," vol. v., "Diseases of the Respiratory Organs." In Juergensen's paper therein, entitled "Hypostatic Processes of the Lungs" (p. 236), I found that I was supported by one writer, he a master-mind (M. Piorry); and the simple fact may now be stated that I had indicated a diseased condition besides those usually recognized by the profession; and this had in a great measure corresponded with what Piorry has pointed out as a distinct form of disease, to be recognized and treated by the general practitioner. I had noticed and had endeavored to attract attention to a most marked and characteristic morbid condition, which physicians in hospital practice must have met, and must now meet, but failed to recognize, name, and separate from others.

Juergensen writes as follows, *loc. cit. sup.*: "Hypostatic pneumonia and hypostatic conditions of the lungs were first recognized as a distinct form of pulmonary disease through the labors of French writers. Pre-eminent among them is Piorry, who handles the subject with great clear-

ness, and whose teachings are based upon a rich experience. He likewise gave the disease its name."

"Piorry proved by experiment that a hypostatic condition diagnosticated during life did not alter its location after death, under the laws of gravitation. As Piorry made his diagnosis long before death, it was evident that this condition did not result during the death-struggle. By means of these experiments *hypostasis ceased to be a condition of but little pathological significance*. If it occurred during life, then it must have an influence on life."

To quote still from Juergensen: "Does an *inflammation of the lung* actually exist? Is the term 'hypostatic pneumonia' correct? Here we must agree with Piorry, who answered this question in the negative in his nomenclature, and afterward still further confirmed this opinion." "He calls this form of disease *pneumonémie hypostatique*, and gives as a synonyme *engouement pulmonaire*" (vol. v., p. 238).

So I am sustained by Piorry, not only as regards the existence of a special disease and in the non-existence of an inflammation of the lung, but also in the use of the identical designation *engouement pulmonaire*, which may be equivalent to "engorgement of the lungs."

Pneumonémie, under Piorry's peculiar phraseology, it will be observed, is not to be confounded with pneumonia; it is equivalent to congestion. Juergensen, in going on to describe "*the conditions which give occasion to the rise of hypostatic infiltrations of the lungs*," makes no difficulty whatever regarding the existence of the condition, but describes its pathological anatomy, symptomatology, etc.

I need only quote the following, which may be compared with my own observations: "The local symptoms of hypostasis demonstrable by physical examination are the following: At first diminished resonance on percussion, beginning at the lower angle of the scapula, and on auscultation a lessening, sometimes almost a cessation of the respiratory murmur, which is vesicular, or may be quite indefinite in character. At the point of attack the vocal fremitus is weak. If hypostasis is complicated with a coexistent catarrh, new features foreign to the former disease will appear. Mucous râles, for example, are usually absent in simple hypostasis. The dulness on percussion and the auscultatory signs, as a rule, extend slowly from below upward. There is a period at which absolutely no breathing is to be heard over the consolidated portion (Piorry). Then mucous râles gradually become audible, those in the larger tubes appearing first. In case of a fatal termination extensive oedema of the lungs supervenes, accompanied by the auscultatory sign peculiar to that condition." I have not been able to confirm the supervention of oedema; certainly never got the crepitant râle, or other signs of oedema, just before death, nor at any period.

However, both of us recognized and marked out a collection of symptoms which are often found associated, but which had not previously been designated as characterizing a special diseased condition. This collection can have no other name than Engorgement of the Lungs; and all such terms as pneumonias, in any form, hyperæmias, oedemas, etc., must be rejected, as I will endeavor to show later on.

The merit of Piorry consists in his freeing hypostatic processes from the imputation—ancient and deep-grounded in all writings—of occurring only just before death; and giving it its true place as a diseased condition of variable duration, to be recognized during life. What he has done we have just read from Juergensen's paper.

If my cases of engorgement of the lungs were proved to be only forms of hypostasis, yet I also recognized them as unconnected with position, or with the death-struggle; as existing, and to be studied and treated long before dissolution. But I do not regard "engorgement of the lungs" as dependent altogether upon hypostasis, therefore my observations do not conform strictly to Piorry's.

Woillez, in his "Dictionnaire de Diagnostique médicale," Paris, 1870, citing "the diseases of the lungs which

can be diagnosticated during life," does not even refer to such a disease; he includes only: Congestion, Pulmonary Apoplexy, Pneumonia, Gangrene of the Lungs, Cœdema, Tubercles (Phthisis); Lardaceous Degeneration, or Amyloid; Cancer, Hydatid or other Tumors, Vesicular Emphysema, Accidental Perforation of the Organ (Pneumothorax).

I quote one passage from his rather elaborate description of "Congestion of the Lungs," to show how far removed this also is from the disease we are considering:

"*Congestion du Poumon : 1° Éléments de la diagnostique*, considérée comme maladie au même titre que la bronchite franche et la pneumonie; l'hypérmie pulmonaire débute subitement par une douleur du côté de la poitrine, avec fièvre le plus souvent légère, parfois intense, une dyspnée très variable, avec absence de toux, ou toux assez rare, sèche ou suivie d'expectoration muqueuse, transparente, parfois un peu teintée de sang."

All he has to say under the head of Engorgement ("Engouement") is as follows: "This word is applied principally to the lungs and to the intestines. It expresses in the first case a state of inflammatory congestion still ill defined." This was in 1870.

Professor John Guitéras, late of this city, informs me that Woillez has recently (1889) written a paper on "The Acute Diseases of the Chest," published in one of the New York medical journals, with six cases of acute fatal congestion of the lungs from exposure to cold.

In view of the claims which I have ventured to make, the burden of proof is upon me to show that engorgement of the lungs as a disease, though not infrequently met with, is not described or recognized by the prominent authors in this country. One or two of these must therefore be examined.

But first, as to the literature of the subject in this country. All that we have is a paper by the present writer (which had wholly passed out of his mind) published in the *American Journal of the Medical Sciences* for October, 1869, being one section¹ of an article entitled "Certain Pathological Conditions."

It is true that the so-called "hypostatic pneumonia" has been referred to in the edition of 1886 of "Flint's Practice," by Flint, Jr., and Welch, but this is a comparatively recent publication. The existence of such a disease is regarded as problematical.

Flint writes as follows: "Passive hyperæmia occurring in the dependent portions of the lungs is called hypostatic

¹This being brief, I insert here, as follows:

Frequency of Serous Engorgement of the Lungs.—I have very frequently met with a condition of the lungs which I have been in the habit of designating in my clinical teaching before students as "serous engorgement," or simply pulmonary engorgement.

The lower lobes on either side are the parts usually implicated. There is some dulness on percussion, some deep-seated respiration, with rough breathing, and at times a little crepitation. Coexisting with such a condition there is neither pneumonia, bronchitis, tuberculosis, phthisis, nor any cavity. The post-mortem examinations have repeatedly disclosed the unmixed character of the pathological state.

It is a result of neglected catarrhs, previously existing bronchitis or pneumonia in a chronic form, and sometimes the engorgement is partly hypostatic; but this term should be reserved for post-mortem changes, or those occurring just before death. I think the term used above can often be safely and properly used as a distinctive one, marking a substantive condition. The term "congestion" should be applied only to active determination of blood to the lungs, as in apoplexy, etc. In "serous engorgement" there is often a large amount of serous fluid mixed with air, which escapes on cutting into the lungs, and the dulness is not absolute when the patient is examined before death, as the lung tissue is still partly permeable to air.

This serous engorgement often also accompanies tuberculosis of the lungs, as, for example, when cavities or granular tubercular matter is found in the upper portion of the organs, the lower and more dependent portions are simply engorged with serum, and furnish corresponding auscultatory and percussional signs. But the morbid alterations referred to as existing at the base or more dependent portions of the lungs are not "tubercular infiltrations," which almost always have their seat under the clavicles.

If "serous engorgement" is one and the same with "oedema," then it exists much more frequently than the books would teach us to believe it does, and Laennec was wrong in stating that the crepitant râle characterized three conditions, namely: The forming stage of pneumonia, the congested tissues around a hemorrhagic spot, and oedema (in which he is correct as far as my experience goes), for in the serous engorgement described above the crepitant râle is rarely if ever heard. In true "oedema" of the lungs consequent upon measles, the crepitant râle is heard over the entire region of the chest.

congestion. The conditions which favor the production of hypostatic congestion are enfeebled heart's action and the maintenance of the body in one position for a long time. It is met with in acute infectious diseases, in the aged, and in the course of chronic diseases which occasion general debility. The higher grades of hypostatic congestion result in a transudation of bloody serum. The condition of lung thus produced has been called splenization. . . .”

Now, our cases are not pneumonias—there is no true splenization, they did not necessarily depend upon acute infectious diseases, did not exist in the aged; the term congestion is not applicable, as I have shown, nor were they dependent upon debility, as before stated. References also to these points have apparently only been made in editions of recent years.

Professor S. C. Chew has an able paper, written in his usual style of excellence, in vol. iii. of Pepper's “System of Medicine,” entitled “Congestion and Ædema of the Lungs (Hypostatic Pneumonia).” What is said below will include all the arguments I have to offer in the effort to show that he also does not include Engorgement of the Lungs in the subject-matters treated by him, and I refrain from a minute analysis only on account of want of space.

The work on “Practice,” third edition, 1885, by so deservedly conspicuous an authority as Dr. Loomis, must also be examined; but having the greatest respect for his character, ability, and experience, I write in no undue critical or controversial spirit. The terms “engorgement,” or “serous engorgement,” are nowhere found, either in the index or in the body of the work; nor is there any other disease referred to by him which can be confounded with “engorgement of the lungs,” as I will endeavor to show by a careful analysis.

The nearest approach thereto will be discovered in the section entitled “Hyperæmia of the Lungs,” which is divided by Loomis into “Active Hyperæmia, or Fluxion, and Passive Hyperæmia, or Pulmonary Congestion.”

With the first, active hyperæmia, our disease, “engorgement of the lungs,” will not be confounded. The second, passive hyperæmia, embraces a subdivision, viz., hypostatic congestion, which somewhat resembles “serous engorgement,” but if the diagnostic differences are closely scanned, the incompatibilities can readily be exposed. In justice to the author we quote as follows (p. 113):

“Passive hyperæmia or pulmonary congestion depends upon an obstruction to the return circulation. It occurs with varying appearances and anatomical characteristics that have led to its subdivision into *splenization*, *brown induration*, and *hypostatic congestion*. A form of active hyperæmia has, because of its physiological cause and situation, been called *compensatory hyperæmia*. Other divisions are sometimes made, but all the varieties can probably be classified under these heads.”

I have explained elsewhere why “engorgement of the lungs” cannot be confounded with *splenization*, with *hypostatic*, or any other active *congestion*; still less is it possible to confound it with “brown induration,” or “compensatory hyperæmia”—this will be admitted without hesitation by everyone. But I must recur to “hypostatic congestion.” This, unless in a modern and changed sense of the term—and Loomis fails to say that views have changed with regard to it—is not to be confounded, as I explained, with “engorgement of the lungs;” unless it is assented to that hypostatic congestion (laying aside for the present any objection to the term *congestion*) is not necessarily associated with the position of the body or with post-mortem changes, as Piorry's researches have proven. It is still a grave misnomer to apply the term hypostatic to a disease where there is no necessary hypostasis. If the characteristics are abandoned which make it a case of hypostasis, then we had better change the name for one more appropriate, and call the disease engorgement of the lungs, which is far the most applicable, as it is descriptive of the true condition. But, finally, our “engorgement of the lungs” is

not *hypostatic congestion!* and Piorry himself called his hypostatic congestion “*engouement pulmonaire*.” He encountered the same diseased condition that I did, and the difficulty was met in each case by the application of analogous terms.

Let us see what the learned author (Loomis) says of “hypostatic congestion”—I quote *verbatim et literatim*: “*Hypostatic* congestion is a term applied to that form of hyperæmia which occurs in the most dependent parts of the lungs; it is usually bilateral in those dying of diseases which have confined them to bed for a long time. It very closely resembles splenization, but the lung-tissue is very friable instead of doughy, and the little whitish or reddish points which are seen in splenization are absent in hypostatic congestion. The lung-texture itself is but little altered. Low forms of pneumonia are liable to occur in hypostatic congested parts of the lung, and hence some call it ‘hypostatic pneumonia,’ and others again call it splenification (differing from the above described splenization).”

Our disease has nothing to do primarily or essentially with hypostasis, nothing to do with being “confined in bed for a long time;” there is also no splenification; it has nothing to do with any form of pneumonia. The crepitant râle, fever, or rusty-colored sputa—one or more—should be essential to every morbid state to which the term pneumonia is applied. I have repeatedly stated that none of these symptoms characterize engorgement of the lungs. Besides, the description by the able pathologist quoted above is entirely too inadequate; there is not enough relating to the morbid anatomy, symptomatology, etc., of hypostatic congestion to prove that it agrees with “engorgement of the lungs,” and hence that the latter is no new disease.

As I have satisfactorily disposed of the subdivisions of “passive hyperæmia,” I might well be dispensed from considering the characteristics of passive hyperæmia itself. But I will state that this is not to be confounded with “engorgement of the lungs.” Loomis gives some of the characteristics of passive hyperæmia by which the identity of the two will be proven to be irreconcilable. For example, he does not mention serum as exuding, but only “dark blood;” “bronchial tubes and pleura show *post-mortem staining*.” “Engorgement of the lungs” has nothing to do necessarily with *post-mortem staining*; it is not dependent upon the position of the body—for a long or a short period; or upon the accidencies of dissolution—upon recumbency or decubitus; it bears no relation whatever to death or the death-struggle—it has, indeed, existed for weeks or months before death.

In “engorgement of the lungs” there is serum and blood mixed—so the term *hyperæmia*, also, would, for this reason, not be comprehensive enough.

Here are some cogent additional reasons: Dr. Loomis, in speaking also of the different varieties of pulmonary hyperæmia (p. 115), says, “blood-stained, watery expectoration is the prominent objective symptom of pulmonary congestion. The advent of *active* hyperæmia is usually very sudden.”

I have already shown that “engorgement of the lungs” is not congestion; and also that it is never active and sudden in its onset—as is congestion. In “engorgement of the lungs” “watery expectoration is not a prominent symptom.” In fact it does not exist in cases of engorgement of the lungs.

Dr. Loomis again repeats (p. 116) that the “diagnosis of pulmonary congestion is not difficult if one considers the circumstances under which it occurs, and the two prominent symptoms, viz., the dyspnea and the copious, watery, blood-stained expectoration.” He also says that “cedema” is characterized by “blood-stained sputum” (p. 116).

These admissions by a pathologist of great experience settle the question that neither of these diseases is synonymous with “engorgement of the lungs,” in that blood-stained sputum has not been seen by myself in the latter,

nor referred to in the statement of the pathology of the diseased condition with which the researches of my old master, Pierry, have been so prominently associated.

Résumé.—The pathological condition referred to should be best designated and known as *Engorgement of the Lungs*:

Because, 1st, *Engorgement of the lungs* may include *blood and serum*, both being invariably present.

2d. Term "*hypostatic*" objectionable; because condition does not depend on hypostasis.

3d. "*Congestion*" objectionable; because congestion is an acute condition (see body of paper).

4th. "*Hyperæmia*" objectionable; because it necessitates the presence of blood, and does not include serum, which is always present.

5th. "*Pneumonia*," or "*pneumonic*," objectionable; because the crepitant râles, fever, rusty-colored sputa, bronchial respiration, and hepatization, are all *absent*.

6th. "*Œdema*" objectionable; because "in œdema the expectoration is always frothy and watery in character and abundant" (Loomis), and there is dyspnoea. These do not occur in engorgement of the lungs, and in the latter there is, also, no blueness of lips, lividity, cold extremities, etc., as are found in extreme cases of œdema.

Illustrative Cases.—During the past year the following (which was a tolerably fair specimen, but the records are meagre) occurred during my attendance upon the Marine Hospital wards:

CASE I.—E. O—, white, aged thirty, admitted August 7, 1888, with a diagnosis of bronchitis. Evening temperature, 100° F.; morning, $98\frac{2}{3}^{\circ}$ F. His condition was as follows: Dulness over front of right lung; left lung resonant; no râles; vocal resonance almost amounting to pectoriloquy in the right lung posteriorly, above the scapula. No indication of phthisis, fibroid phthisis, bronchitis, or pleurisy.

Diagnosis.—“Engorgement of the lungs.”

Note.—Similar symptoms naturally occurred in a man who died from drowning a few days previously.

August 11th.—Revulsives were employed; much less dulness observed at the base of right lung anteriorly and posteriorly; temperature ranges from 97° to 101° F. From physicians present, when invited to express their opinions based on a consideration of the symptoms, the replies varied, as follows: “Incipient phthisis,” “Catarrhal pneumonia,” “Hypostatic congestion,” “Œdema,” etc.

There had been no evidence of catarrhal symptoms or pneumonia, no crepitant râles, rusty-colored sputa, etc. Hypostatic congestion was the closest approach; but this, as we then interpreted it here, is a condition characterizing the very latest ante-mortem state, results from a protracted recumbency upon the back, occurs in the aged or when the powers of life are enfeebled. It was not congestion of the lungs, for attacks of this are sudden and violent, and there is active hyperæmia.

The next case is extracted *verbatim* from my notes, under the date 1879, and therein entitled “serous engorgement,” which term was used here as synonymous with “engorgement of the lungs.”

CASE II.—Woman, colored, aged fifty, entered hospital October 5, 1879. Complained of some derangement of chest and stomach; no active disturbance of stomach; enlarged liver, which was hard; no fever or dyspnea; cough, but no expectoration; dulness over heart; resonance over apices of both lungs, but at back of right and left lung partial dulness, with subcrepitant râles, especially in right lung; dulness more marked at inferior margin of lungs.

Diagnosis.—“Serous engorgement;” possibly water in pericardium, but no pleuritis.

Death occurred rather suddenly—subject complaining of oppression of the chest; pulse normal. All the post-mortem phenomena corresponded with the diagnosis.

Autopsy.—Above thoroughly sustained, as follows: Fluid in pericardium; liver large, hard, thickened; spleen medium size. Right lung engorged, and poured out serum when cut; dilatation of right ventricle and general enlargement of the heart, no special valvular disease; veins of right side of the neck enlarged—the jugular was more than an inch in diameter.

Note, 1879.—I make a distinction between “engorgement,” congestion, and also œdema—the justness of which this case confirmed. Drs. Rhett, Andrews, and Wanamaker, house physicians, were present.

CASE III.—A. K—, colored, aged twenty-five, admitted May 22, 1889. Had had pneumonia; base of both lungs dull anteriorly and posteriorly; no swelling, no râles.

May 23d.—Same condition as on previous day; no fever, no râles; base of both lungs dull, but not absolutely, as would be if there was water on the chest; no pleurisy, no bronchitis.

May 28th.—Examined. No fever; back of both lungs dull, dulness also extending in front; respiration not quickened; no râles, cough, or pleurisy. So:

Diagnosis.—Engorgement of lungs.

This case presented also a rare example, which I have recently seen repeated, of greatly diminished respiration, without quick breathing. Entered hospital with a diagnosis of “pneumonia of right lower lobe.” If so, the pneumonia is well, and is followed by serous engorgement.

CASE IV.—E. J—, admitted February 14, 1877, aged twenty-two. Had spitting of blood; no dulness under either clavicle, and anterior portions of both sides of chest resonant, respiration loud. Puerile respiration at back of left lung; percussion dulness over back of right lung, extending to diaphragm and liver, and absence of respiration; subcrepitant râles posteriorly.

Diagnosis.—Serous engorgement of right lung; spleen somewhat enlarged; respiration not quickened; pulse 120; temperature, 100° F. Ordered, li. Calomel, gr. $\frac{1}{2}$; digitalis, gr. j.; ipecac, gr. $\frac{1}{2}$; squill, gr. ij. in each pill. One t.i.d.; chest to be rubbed with iodine and mercurial ointment (U. S. D.), and cod-liver oil and whiskey, on account of weakness.

February 25th.—Still weak. Subcrepitant râles very evident over right lung, with dulness—the serous engorgement involves whole of right lung. Dulness exists still at back of right lung also—not so marked as formerly, and crepitation on inspiration also diminished. Applied emplast. cantharides, 4 by 5, back of right lung.

Died April 3, 1877.

CASE V.—P. B—, admitted 1888. Engorgement of lungs (or hypostatic congestion); had been in bed for three weeks on his back; no râles, partial dulness; all signs of hypostatic engorgement, viz., impaired respiration, posterior portion of lower lobe of lungs, mostly base and centre, dull.

Left hospital September 24th completely well. Took stimulants, milk diet, etc. In the above case “hypostatic congestion” and “engorgement of the lungs” are convertible terms.

CASE VI.—P. M—, colored, aged sixty; admitted September 7, 1888, with a diagnosis of “chronic gastritis and malaria with anæmia.”

September 8th.—Examined. Physical signs: Dulness on right side of chest, anteriorly, extending up to fifth intercostal space, and to about the seventh posteriorly; respiration in this region not good; left lung healthy; heart-sounds normal, but somewhat labored.

A tumor, about four inches in diameter and hard to the touch, found in the region of the stomach, just below the ensiform cartilage; no pulsations, but with considerable constriction of the abdominal walls just above the umbilicus—supposed cancer of stomach! Patient, about eight to ten hours after meals, vomits a considerable quantity of partially digested blood, or black vomit; pain considerable in the region of the tumor, which had been observed for one month past; patient anæmic. Dulness

and impaired respiration extend to the rear of the right lung; probably "engorgement of the lung."

Two cases may be added; one of "acute engorgement of the lungs," with recovery, and one, for comparison, an example of acute *congestion* of the lungs, with an autopsy.

CASE VII. *Acute Engorgement of the Lungs* (from notes, 1886).—D. G.—, white, aged eight; suffered apparently from acute engorgement of the lungs, without crepitant râles; rusty-colored sputa, etc.; a "click" and dulness were present at the back of the right lung, middle region. Recovery took place after eight days' treatment. Temperature 101° to 102° F.; on the fifth day it was 103° F., even after using the following mixture: Rx. Sirup of squills, ipecac, and soda with nitre. This combination failed. Then the following: Rx. Calomel, gr. $\frac{1}{4}$; Dover's powder, gr. $\frac{1}{2}$; soda, gr. j. Six powders, each containing the above. S.: One every three or four hours. Did not resolve the inflammation. Then local applications of mustard, followed by a thapsia plaster, with flaxseed tea as a drink. As fever became higher, employed, successively: Rx. Muriate of ammonia, 3 j.; carb. of ammonia, 3 j.; acetate of potash, 3 ij.; tinct. aconite, gtt. xx.; tinct. sanguinaria, 3 iij.; fluid extr. licorice, 3 ij.; water to $\frac{1}{2}$ iv. S.: Teaspoonful every two hours. Gave also a stimulating diaphoretic tea, made with senega and serpentaria, t.i.d.

Before the last mentioned was given, had used also, ineffectually, the following sedative and relaxant expectorant: Rx. Wine of antimony, 3 iij.; sirup squills, sirup senega, $\frac{1}{2}$ ss.; tinct. digitalis, 3 j.; sirup wild cherry bark, $\frac{1}{2}$ j.; tinct. lobelia, 3 iij.; water to $\frac{1}{2}$ iv. S.: Teaspoonful, t.i.d., and oftener if cough is troublesome.

He never had much expectoration, but his cough was troublesome, with constant fever; no pain; pulse, always 120, and respiration, 28. Under the aconite mixture, which is an excellent combination for pneumonic (?) engorgement, the temperature went down to 97° F. To day, March 16th, sitting up; temperature, $98\frac{1}{2}$ F. On the fifth day I used an oil-silk jacket over his flannel; the latter was wet with perspiration every night, and was changed.

It is instructive to compare a case like the above with our cases of "engorgement of the lungs." In the latter, which is not acute, there is no such high temperature. The absence of the crepitant râle, rusty-colored sputa, etc., in the above case, make its relations to pneumonia doubtful. Therefore I consider it a case of acute engorgement—not congestion, not pneumonia.

CASE VIII. *Congestion of Lungs; with an Autopsy* (from notes, December 5, 1880).—This case is introduced for comparison with cases of "engorgement of the lungs" and to show the acute nature and rapid invasion of congestion.

S. A.—, white, aged twenty-five, entered City Hospital, July 3, 1880, two days before death. Not very ill, apparently; said to have intermittent fever, and that on the previous night fever was very high. I could not ascertain that he had any congestive chills, or any prominent symptoms, after close inquiry from nurses and those lying near him.

When I saw him, July 4th, at 9 A.M., his respiration was slow and oppressed, and gradually becoming more so. He died in ten minutes after, though his pulse was not feeble—explained by the fact that death was caused by an apoplectic invasion of the lungs, while the heart was less involved.

Autopsy, July 5th. The cavities of the heart contained some blood; the pericardial sac held much bloody serum; much blood free in cavity of the thorax—a quart or more; both lungs hepatized or black with blood; blood was pouring from his nose at the autopsy (there had been white froth upon his lips at death, in striking contrast with his dark face). The spleen was soft and friable—not large, four by five inches; liver enlarged.

This man had not been severely purged (colliquative diarrhoea, whether natural or artificial, being, in my opin-

ion, a frequent cause of congestive chills from diminished power of reaction which they give rise to). He must have died after brief sickness of *congestion* of the lungs—this being the most decided post-mortem confirmation of such a diagnosis I have ever seen. Have never seen so bloody a lungs and thorax; the structure of the heart also seemed softened and infiltrated with blood.

I cannot discover that this man had been purged before or after his admission. It is stated by the house physician that his fever had been very high the previous evening; no testimony showing the existence of any cold, chills, or excessive sweating. I regard diarrhoea, and *high temperature*, which liquefies the blood, as the dangerous symptoms. He had had ten grains or more of quinine, given him t.i.d. since his entrance into hospital.

JABORANDI AND PILOCARPINE IN BRIGHT'S DISEASE.

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THE leaves of *Pilocarpus pinnatus* have been used to produce sudorific effects for centuries. The alkaloid has been before the profession for the last twelve years. Nevertheless, the truth of a recent statement will be readily admitted. Willoughby says (*The Lancet*, May 25, 1889): "There are few drugs of which the physiological action is so direct and palpable, but of which the therapeutic uses are so undetermined."

In the discussion which followed the reading of Dr. C. S. Wood's paper on "Some Points in the Treatment of Chronic Albuminuria, or Bright's Disease" (THE MEDICAL RECORD, March 24, 1888), pilocarpine was mentioned by but one speaker. Dr. Page's faith in the drug was not of a very exalted nature. "Pilocarpine," he added, "has been recommended for the purpose of eliminating this so-called uremic poison. The chief objections to this remedy are its depressing effect upon the heart, and sometimes it causes such a profuse salivary secretion that the patient almost strangles." So, too, Heither, of Vienna: "He had never seen any good result follow the use of pilocarpine in renal disease after ascites had once set in. He admitted that it produced its physiological effects of increased perspiration and urination, but believed these to be purely temporary, and to have no beneficial result; whereas the depression consequent upon the use of the drug was not seldom alarming."

Before the New York Academy of Medicine (*Birmingham Review*) Dr. Francis Delafield brought forward a paper on the "Treatment of Acute and Subacute Nephritis" (THE MEDICAL RECORD, March 23, 1889). It was discussed by seven representative physicians of that great medical centre. But one referred to the use of pilocarpine—in truth, the remedy was almost ignored.

On the other hand, the general consensus of recent authorities on *materia medica* is that expressed by Stillé ("National Dispensatory"): "When dropsy arises in connection with desquamative tubular nephritis (especially scarlatinous), or even with interstitial nephritis, the medicine is very efficient; in the former case often leading to a cure, in the latter to a prolongation of life."

From Professor Wagner's clinic at Buda-Pesth comes the following: "When used according to the above-mentioned principles (*quod vide*), pilocarpine will be found in most cases of Bright's disease, even when hot baths and other diaphoretics prove useless, always to diminish dropsy to such an extent that the patient is more or less protected against dangerous uremic suffocative attacks. In this way it may be possible to obtain a relative cure; that is, in secondary granular contracted kidney" (*British Medical Journal*).

In *The Lancet* for December 22, 1888, J. G. Marshall, B.A., M.B. Cantab., writes: "The value of jaborandi and its derivatives in the treatment of the dropsy of Bright's disease cannot be overestimated. By its use I have re-